Symptoms and notes on heavy metal toxins

Human beings have been exposed to heavy metal toxins for an immeasurable amount of time. The industrialization of the world has dramatically increased the overall environmental 'load' of heavy metal toxins to the point that our societies are dependent upon them for proper functioning. Industry and commercial processes have actively mined refined manufactured burned and manipulated heavy metal compounds for a number of reasons. Today heavy metals are abundant in our drinking water air and soil due to our increased use of these compounds. They are present in virtually every area of modern consumerism from construction materials to cosmetics medicines to processed foods fuel sources to agents of destruction appliances to personal care products. It is very difficult for anyone to avoid exposure to any of the many harmful heavy metals that are so prevalent in our environment. While it does not appear that we are going to neutralize the threat of heavy metal toxicity in our communities nor decrease our utilization of the many commercial goods that they help produce we can take steps to understand this threat and put into action policies of prevention and treatment that may help to lessen the negative impact that these agents have on human health.

Heavy metal toxins contribute to a variety of adverse health effects. There exist over 20 different heavy metal toxins that can impact human health and each toxin will produce different behavioral physiological and cognitive changes in an exposed individual. The degree to which a system organ tissue or cell is affected by a heavy metal toxin depends on the toxin itself and the individual's degree of exposure to the toxin. Here are presented just 5 of the many hazardous metal toxins that are commonly encountered by humans. Each of these metals affects an individual in such a way that its respective accumulation within the body leads to a decline in the mental cognitive and physical health of the individual. The specific sources of exposure where the metals tend to be deposited and the adverse health effects of each metal are identified below.

1. Aluminum (CAS# 7429-90-5) Sources of exposure: Aluminum is a naturally occurring metal that has been utilized by humans for a number of years. It is the third most abundant element in the earth's crust (approximately 8% of the crust is composed of aluminum compounds) and is apparent is small quantities (from 3-2400 ppb) in seawater (Venugopal and Luckey 1978). Incidences of acid rain on the planet have increased the availability of aluminum to various biological systems. Acid rain is able to dissolve aluminum compounds that are naturally found in soil and rock thus increasing their prevalence in soils and fresh and salt-water sources. Because of this aluminum concentrations can be seen in various fresh and salt-water marine lives and in plants that have been grown in aluminum laden soil. Humans have processed aluminum compounds for years and its use is apparent in many different forms of industry. Because of its many industrial and commercial uses aluminum is consumed and/or handled by many individuals on a daily basis. Today aluminum can be found in cookware aluminum foil dental cements dentures leather tanning preparations antacids antiperspirants appliances baking powder buffered aspirin building materials canned acidic foods food additives lipsticks construction materials (the automotive aviation and electrical industries all use aluminum compounds for various uses) prescription and over-the-counter drugs (anti-diarrhea agents hemorrhoid medications vaginal douches) dialysates vaccines processed cheese paints toothpaste fireworks and "softened" and normal tap water (ATSDR 1990 Wills and Savory 1985). Aluminum has been found in at least 489 of the 1416 (34%) National Priorities List (NPL) sites identified by the Environmental Protection Agency (EPA) (ATSDR 1995).

Target tissues: Aluminum accumulates in the brain muscles liver lungs bones kidneys skin reproductive organs and stomach (ATSDR 1990 Wills and Savory 1985). Depending on the source of exposure aluminum can be absorbed through the gastrointestinal (GI) tract or the lungs. Absorption through the GI tract is slow due primarily to pH factors but once absorbed it is distributed to the bones liver testes brain and soft tissues. Following aluminum inhalation deposition occurs primarily within the lungs (Venugopal and Luckey 1978).

Signs and Symptoms: Aluminum toxicity can produce a number of clinical signs and symptoms.

Common are excessive headaches abnormal heart rhythm depression numbness of the hands and feet and blurred vision (Kilburn and Warshaw 1993). Aluminum toxicity has been shown to produce impairment in choice reaction time long-term memory psychomotor speed and recall in affected individuals as compared to controls (Wills and Savory 1985). Animal studies have shown similar impairment in locomotors activity/response and spatial learning in rats receiving dietary aluminum for a period of 12 weeks (Commissaris et al. 1982). In a study conducted with patients receiving dialysis for renal failure aluminum was believed to be a causal agent in the development of dialysis encephalopathy (or "dialysis dementia") a special form of bone disease known as osteomalacic dialysis osteodystrophy and anemia (Wills and Savory 1985). In this study individuals had been receiving concentrations of aluminum directly from their dialysate. Similarly long-term hemo-dialysis patients have exhibited a progressive neurological syndrome that includes speech disorders dementia myoclonus and encephalopathy (Perl and Brody 1980). Evidence suggests that inhaled aluminum may contribute to the development of pulmonary fibrosis and to a lesser degree pulmonary granulomatosis (ATSDR 1990). Aluminum may be involved in a myriad of neurodegenerative diseases. Dr. McLaughlin MD F.R.C.P. a professor of physiology and medicine and the director of the Centre for Research in Neurodegenerative Diseases at the University of Toronto states: "Concentrations of aluminum that are toxic to many biochemical processes are found in at least ten human neurological conditions"(Crapper-McLachlan 1980). Recent studies suggest that aluminum may be involved in the progression of Alzheimer's Disease Parkinson's disease Guam ALS-PD complex "Dialysis dementia" Amyotrophic Lateral Sclerosis (ALS) senile and presenile dementia neurofibrillary tangles clumsiness of movements staggering when walking and an inability to pronounce words properly (Berkum 1986; Goyer 1991; Shore and Wyatt 1983). To date however we do not completely understand the role that aluminum plays in the progression of such human degenerative syndromes. Chronic aluminum exposure has contributed directly to hepatic failure renal failure and dementia (Arieff et al. 1979). Other symptoms that have been observed in individuals with high internal concentrations of aluminum are colic convulsions esophagitis gastroenteritis kidney damage liver dysfunction loss of appetite loss of balance muscle pain psychosis shortness of breath weakness and fatigue (ATSDR 1990). Behavioral difficulties among schoolchildren have also been correlated with elevated levels of aluminum and other neuro-toxic heavy metals (Goyer 1991). And aluminum toxicity may also cause birth defects in new-borns (ATSDR 1990).

Medical tests for aluminum screening: Blood urine feces hair and fingernails.

2. Arsenic (CAS# 7440-38-2) Sources of exposure: The use of this toxic element in numerous industrial processes has resulted in its presence in many biological and ecological systems. Ground surface and drinking water are susceptible to arsenic poisoning from the use of arsenic in smelting refining galvanizing and power plants; environmental contaminants like pesticides herbicides insecticides fungicides desiccants wood preservatives and animal feed additives; and human made hazardous waste sites chemical wastes and antibiotics. Arsenic concentrations are apparent in the air as a result of the burning of arsenic containing materials such as wood coal metal alloys and arsenic waste (ATSDR 1989; Morton and Caron 1989). Arsenic concentrations can also be found in specialty glass defoliants marine life (primarily fish and shellfish) and riot-control gas (Hine et al. 1977). Arsenic is present in at least 781 of the 1300 (60%) NPL sites as identified by the EPA (RAIS 1992).

Target tissues: Many arsenic compounds are readily absorbed through the GI tract when delivered orally in humans. Absorption within the lungs is dependent upon the size of the arsenic compound and it is believed that much of the inhaled arsenic is later absorbed through the stomach after (respiratory) mucocillary clearance (ATSDR 1989). After the absorption of arsenic compounds the primary areas of distribution are the liver kidneys lung spleen aorta and skin. Arsenic compounds are also readily deposited in the hair and nails (U.S. EPA 1984).

Signs and Symptoms: Arsenic is a highly toxic element that has been used historically for purposes of suicide and homicide. Its health effects are well known and multiform. Acute exposure to arsenic compounds can cause nausea anorexia vomiting abdominal pain muscle

cramps diarrhea and burning of the mouth and throat (ATSDR 1989). Garlic-like breath malaise and fatigue have also been seen in individuals exposed to an acute dose of arsenic while contact dermatitis skin lesions and skin irritation are seen in individuals whom come into direct tactile contact with arsenic compounds (Feldman et al. 1979). A large acute oral dose has caused tachycardia acute encephalopathy congestive heart failure stupor convulsions paralysis coma and even death (Morton and Caron 1989). Animal studies have shown similar acute effects when arsenic compounds were delivered orally to Rhesus monkeys (Heywood and Sortwell 1979). Repeat exposure to arsenic compounds have been shown to lead to the development of peripheral neuropathy encephalopathy cardiovascular distress peripheral vascular disease EEG abnormalities Raynaud's phenomenon gangrene of the lower legs ("Black foot disease") acrocyanosis increased vasopastic reactivity in the fingers kidney and liver damage hypertension myocardial infarction anemia and leukopenia (ATSDR 1989; Blom et al. 1985; Feldman et al. 1979; Heyman et al. 1956; Hine et al. 1977; Langerkvist et al. 1986; Morton and Caron 1989). Other chronic effects of arsenic intoxication are skin abnormalities (darkening of the skin and the appearance of small "corns" or "warts" on the palms soles and torso) neurotoxic effects chronic respiratory diseases (pharyngitits laryngitis pulmonary insufficiency) neurological disorders dementia cognitive impairment hearing loss and cardiovascular disease (Blom et al. 1985; Kyle and Pease 1965; Morton and Caron 1989). A significantly higher percentage of spontaneous abortions have been shown in a population living near a copper smelting plant; lower birth weights of babies born to this same population are seen and an abnormal percentage of male to female births is also apparent suggesting that arsenic affects babies in uterus (Nordstrom et al. 1979). Studies have shown close associations between both inhaled and ingested arsenic and cancer rates. Cancers of the skin liver respiratory tract and gastrointestinal tract are well documented in regards to arsenic exposure (IARC 1980; Lee-Feldstein 1989). Several arsenic compounds have been classified by the US Environmental Protection Agency as a Class A-Human Carcinogen (IARC 1987).

Medical test for arsenic screening: Urine (best) hair and fingernails.

3. Copper (CAS# 7440-50-8) Sources of exposure: Copper occurs naturally in elemental form and as a component of many different compounds. The most toxic form of copper is thought to be that in the divalent state cupric (Cu2+). Because of its high electrical conductivity copper is used extensively in the manufacturing of electrical equipment and different metallic alloys. Copper is released into the environment primarily through mining sewage treatment plants solid waste disposal welding and electroplating processes electrical wiring materials plumbing supplies (pipes faucets braces and various forms of tubing) and agricultural processes (ATSDR 1990a). It is present in the air and water due to natural discharges like volcanic eruptions and windblown dust. Drinking water sources become contaminated with copper primarily because of its use in many different types of plumbing supplies. It is a common component of fungicides and algaecides and agricultural use of copper for these purposes can result in its presence in soil ground water farm animals (grazing animals like cows horses etc.) and many forms of produce (ATSDR 1990a). Copper is also present in ceramics jewelry monies (coins) and pyrotechnics (ACGIH 1986). Though copper is an essential trace element required by the body for normal physiological processes increased exposure to copper containing substances can result in copper toxicity and a wide variety of complications.

Target tissues: Absorption of copper occurs through the lungs gastrointestinal tract and skin (U.S. EPA 1987). The degree to which copper is absorbed in the gastrointestinal tract largely depends upon its chemical state and the presence of other compounds like zinc (U.S.A.F. 1990). Once absorbed copper is distributed primarily to the liver kidneys spleen heart lungs stomach intestines nails and hair. Individuals with copper toxicity show an abnormally high level of copper in the liver kidneys brain eyes and bones (ATSDR 1990a).

Signs and symptoms: Acute toxicity of ingested copper is characterized by abdominal pain diarrhea vomiting tachycardia and a metallic taste in the mouth. Continued ingestion of copper compounds can cause cirrhosis and other debilitating liver conditions (Mueller-Hoecker et al.

1989). Inhaled copper dust or fumes can produce eye and respiratory tract irritation headaches vertigo drowsiness chills fever aching muscles and discoloration of the skin and hair in humans (U.S.A.F. 1990). Vineyard workers exposed to copper fumes for a long period of time developed pulmonary fibrosis and granulomas of the lungs liver impairment and liver disease (cirrhosis fibrosis and various morphological changes). Similar results were obtained in animals chronically exposed to copper containing dust and fumes (Johansson et al. 1984; Stockinger 1981). Further animal studies on copper toxicity have shown varying degrees of liver and kidney damage (necrosis of the kidney; sclerosis necrosis and cirrhosis of the liver) decreased total weight brain weight and red blood cell count increased platelet counts and the presence of gastric ulcers (Kline et al. 1977; Rana and Kumar 1978). Copper also appears to affect reproduction and development in humans and animals. Offspring of hamsters that received copper sulfate injections while pregnant exhibited increased incidences of hernias encephalopathy abnormal spinal curvature and spine bifida (Ferm and Hanlon 1974). Sperm motility also appears to be compromised by the presence of copper in human spermatozoa (Battersby and Morton 1982).

Chronic exposure to copper can produce numerous physiological and behavioral disturbances. Copper toxicity has been characterized in patients with Wilson's Disease a genetic disorder that causes an abnormal accumulation of copper in body tissue. Wilson's disease is fatal unless treated in time. Manifestations of Wilson's Disease include brain damage and progressive demylination psychiatric disturbances; depression suicidal tendencies and aggressive behavior; hemolytic anemia cirrhosis of the liver motor dysfunction and corneal opacities (ATSDR 1990a; Goyer 1991a; U.S. EPA 1987). Some patients may also experience poor coordination tremors disturbed gait muscle rigidity and myocardial infarction (ATSDR 1990a).

Medical tests for copper screening: Blood urine and hair.

4. Lead (CAS# 7439-92-1) Sources of exposure: Lead is the 5th most utilized metal in the U.S. It is mined extensively in Missouri Colorado Idaho and Utah and is used for the production of ammunition bearing metals brass materials solder ballasts tubes containers gasoline products ceramics and weights (ATSDR 1993). Human exposure to lead occurs primarily through drinking water airborne lead-containing particulates and lead-based paints. Several industrial processes create lead dust/fumes resulting in its presence in the air. Mining smelting and manufacturing processes the burning of fossil fuels (especially lead-based gasoline) and municipal waste and incorrect removal of lead-based paint results in airborne lead concentrations. After lead is airborne for a period of ten days it falls to the ground and becomes distributed in soils and water sources (fresh and salt water surface and well water and drinking water). However the primary source of lead in drinking water is from lead-based plumbing materials (U.S. EPA 1989). The corrosion of such materials will lead to increased concentrations of lead in municipal drinking water. Lead from water and airborne sources have been shown to accumulate in agricultural areas leading to increased concentrations in agricultural produce and farm animals (ATSDR 1993). Cigarette smoke is also a significant source of lead exposure; people whom smoke tobacco or breathe in tobacco smoke may be exposed to higher levels of lead than people whom are not exposed to cigarette smoke (RAIS 1994).

Target tissues: Lead is absorbed into the body following inhalation or ingestion. Children absorb lead much more efficiently than adults do after exposure and ingested lead is more readily absorbed in a fasting individual (U.S.EPA 1986). Over 90% of inhaled lead is absorbed directly into the blood. After lead is absorbed into the body it circulates in the blood stream and distributes primarily in the soft tissues (kidneys brain and muscle) and bone. Adults distribute about 95% of their total body lead to their bones while children distribute about 73% of their total body lead to their bones (U.S. EPA 1986a).

Signs and Symptoms: Lead is one of the most toxic elements naturally occurring on Earth. High concentrations of lead can cause irreversible brain damage (encephalopathy) seizure coma and death if not treated immediately (U.S. EPA 1986). The Central Nervous System (CNS) becomes severely damaged at blood lead concentrations starting at 40mcg/dL causing a reduction in nerve

conduction velocities and neuritis (ATSDR 1993). Neuropsychological impairment has been shown to occur in individuals exposed to moderate levels of lead. Evidence suggests that lead may cause fatigue irritability information processing difficulties memory problems a reduction in sensory and motor reaction times decision making impairment and lapses in concentration (Ehle and McKee 1990). At blood concentrations above 70 mcg/dL lead has been shown to cause anemia characterized by a reduction in hemoglobin levels and erythropoiesis-- a shortened life span of red blood cells (Goyer 1988; US EPA 1986a). In adults lead is very detrimental to the cardiovascular system. Occupationally exposed individuals tend to have higher blood pressure than normal controls (Pocock et al. 1984; Harlan et al. 1985; Landis and Flegal 1988) and are at an increased risk for cardiovascular disease myocardial infarction and stroke (US EPA 1990). The kidneys are targets of lead toxicity and prone to impairment at moderate to high levels of lead concentrations. Kidney disease both acute and chronic nephropathy is a characteristic of lead toxicity (Goyer 1988). Kidney impairment can be seen in morphological changes in the kidney epithelium increases in the excretion rates of many different compounds reductions in glomerular filtration rate progressive glomerular arterial and arteriolar sclerosis and an altered plasma albumin ratio (Goyer 1985 1988; Landigran 1989). Chronic nephropathy has lead to increased death rates among occupationally exposed individuals as compared to controls in studies by Selevan et al. (1975) and Cooper et al. (1985). Other signs/symptoms of lead toxicity include gastrointestinal disturbances-abdominal pain cramps constipation anorexia and weight loss-immunosuppression and slight liver impairment (ATSDR 1993; US EPA 1986a).

Children are susceptible to the most damaging effects of lead toxicity. Ample literature shows just how damaging lead is to children. Prenatal and postnatal developments are compromised significantly by the presence of lead in the body. At blood lead concentrations of 80-100 mcg/dL severe encephalopathy occurs. Those children who survive lead-induced encephalopathy typically suffer permanent brain damage marked by mental retardation and numerous behavioral impairments. These children also suffer slower neural conduction velocities peripheral neuropathy cognitive impairment and personality disorders (US EPA 1986a). Tuthill (1996) has found that hair lead levels in children were positively correlated with attention-deficit and hyperactive behavior. Numerous studies have implicated lead as a causal agent in the deterioration of cognitive functioning in children. Studies by Schroeder and Hawk (1986) Burchfield et al. (1980) Otto et al. (1981 1982) and Munoz et al. (1993) have shown IQ deficits in children with blood lead concentrations from 6-70 mcg/dL. Longitudinal studies have given further evidence that lead affects intelligence in exposed children. Studies by Vimpani et al. (1989) McMichael et al. (1988) and Wigg et al. (1988) have shown decreased performance on intelligence tests in lead exposed school children. One study has correlated lower socio-economic status with childhood lead poisoning 50 years after lead exposure (White et al. 1993). Maternal blood lead concentrations and prenatal lead exposure appear to be strong predictors of cognitive performance in offspring. Prenatal exposure may also cause birth defects miscarriage spontaneous abortion and underdeveloped babies (Goyer 1988; McMichael et al. 1988; US EPA 1986d). Lead not only appears to affect cognitive development of young children but also other areas of neuropsychological function. Young children exposed to lead may exhibit mental retardation learning difficulties shortened attention spans (ADHD) increased behavioral problems (aggressive behaviors) and reduced physical growth (Bellinger D. et al. 1990 1992). Lead has been determined by many health experts to be the #1 threat to developing children in our industrial societies.

Medical test for lead screening: Blood urine and hair.

5. Mercury (CAS#7439-97-6) Sources of exposure: Mercury occurs primarily in two forms: organic mercury and inorganic mercury. Inorganic mercury occurs when elemental mercury is combined with chlorine sulfur or oxygen. Inorganic mercury and elemental mercury are both toxins that can produce a wide range of adverse health affects. Inorganic mercury is used in thermometers barometers dental fillings batteries electrical wiring and switches fluorescent light bulbs pesticides fungicides vaccines paint skin-tightening creams vapors from spills antiseptic creams pharmaceutical drugs and ointments (ATSDR 1989a). Inorganic mercury vapor is at high

concentrations near chlorine-alkali plants smelters municipal incinerators and sewage treatment plants. The organic form occurs when mercury is combined with carbon. The most common form of organic mercury is methyl mercury which is produced primarily by small organisms in water and soil when they are exposed to inorganic mercury. Humans also have the ability to convert inorganic mercury to an organic form once it has become absorbed into the bloodstream. Organic mercury is known to bioaccumulate -- or passes up the food chain due an organism's inability to process and eliminate it. It is found primarily in marine life (fish) and can often be found in produce and farm animals processed grains and dairy products and surface salt- and fresh water sources (ATSDR 1989a; Brenner and Snyder 1980). Occupational exposure to mercury containing compounds presents a significant health risk to individuals. Dentists, painters, fisherman, electricians, pharmaceutical/laboratories workers, farmers, factory workers, miners, chemists and beauticians are just some of the professions chronically exposed to mercury compounds.

Target tissues: The absorption and distribution of mercury compounds depends largely upon its chemical state. Organic mercury compounds are absorbed from the gastrointestinal tract more readily than inorganic mercury compounds with the latter being very poorly absorbed. After absorption in the gastrointestinal tract organic mercury is readily distributed throughout the body but tends to concentrate in the brain and kidneys (Goyer 1991b). Approximately 80% of mercury vapor is absorbed directly through the lungs and distributed primarily to the CNS and the kidneys (Friberg and Nordberg 1973). Inorganic and organic forms of mercury have also been seen in the red blood cells liver muscle tissue and gall bladder (Peterson et al. 1991 Dutczak et al. 1991 ATSDR 1989a).

Signs and symptoms: Mercury exposure can result in a wide variety of human health conditions. The degree of impairment and the clinical manifestations that accompany mercury exposure largely depend upon its chemical state and the route of exposure. While inorganic mercury compounds are considered less toxic than organic mercury compounds (primarily due to difficulties in absorption) inorganic mercury that is absorbed is readily converted to an organic form by physiological processes in the liver.

The acute ingestion of inorganic mercury salts may cause gastrointestinal disorders such as abdominal pain vomiting diarrhea and hemorrhage (ATSD 1989a). Repeated and prolonged exposure has resulted in severe disturbances in the central nervous system gastrointestinal tract kidneys and liver. Davis et al. (1974) reported dementia colitis and renal failure in individuals chronically poisoned due to the ingestion of an inorganic mercury containing laxative. Inhaled inorganic mercury can cause a wide range of clinical complications in individuals including corrosive bronchitis interstitial pneumonitis renal disorders fatigue insomnia loss of memory excitability chest pains impairment of pulmonary function and gingivitis (Goyer 1991b ATSDR 1989a). Chronic inhalation of inorganic mercury compounds may result in a reduction of sensory and motor nerve function depression visual and/or auditory hallucinations muscular tremors sleep disorders alterations in autonomic function (heart rate blood pressure reflexes) impaired visuomotor coordination speech disorders dementia coma and death (Clarkson 1989; Goyer 1991b; Fawyer et al. 1983; Piikivi and Hanninen 1989; and Ngim et al. 1992). Ngim et al. (1992) have shown that a group of dentists exposed to mercury vapors occupationally perform significantly worse in neurobehavioral tests that measure motor speed visual scanning visuomotor coordination and concentration verbal memory and visual memory. Kishi et al. (1993) have found that smelter workers exposed to inorganic mercury compounds continue to experience neurological symptoms-tremors headaches slurred speech-senile symptoms and diminished mental capacities eighteen years after the cessation of mercury exposure.

Our understanding of the effects of methyl mercury poisoning comes primarily from epidemic poisonings in Iraq and Japan. In Iraq more than 6000 individuals were hospitalized and 459 died as a result of methyl mercury poisoning. Adults experienced symptoms including parasthesia visual disorders ataxia fatigue tremor hearing disorders (deafness) and coma (Bakir et al. 1973; Mottet Shaw and Burbacher 1985). Neuropathologic observations of exposed individuals have

shown irreversible brain damage including neuronal necrosis cerebral edema gliosis and cerebral atrophy (Mottet Shaw and Burbacher 1985). Iraqi children poisoned through the consumption of methyl mercury containing food products (grains treated with mercury containing fungicides) exhibited nervous system impairment visual and auditory disorders weakness marked motor and cognitive impairment and emotional disturbances (Bakir et al. 1973; Bakir et al. 1978). Individuals in Japan experienced many of these same symptoms after the ingestion of fish containing large amounts of methyl mercury. Similarly autopsies conducted on deceased Japanese in the Minamata Bay have shown pronounced brain lesions cerebral atrophy edema and gliosis in the deeper fissures (sulci) of the brain such as in the visual cortex (Takeuchi 1968). The Japan and Iraq epidemics have clearly established mercury as an agent that can disrupt developmental processes in the unborn and infantile individual. Methyl mercury can pass through the placental barrier and produce many deleterious effects on the unborn fetus (Mottet Shaw and Burbacher 1985). Children born to mercury poisoned mothers were of smaller total weight had decreased brain weights at birth had fewer nerve cells in the cerebral cortex and experienced an abnormal pattern of neuronal migration (Choi et al. 1978; Takeuchi 1968 Amin-Zake et al. 1974). Of those children that survived the epidemic many experienced severe developmental effects like impaired motor and mental function hearing loss and blindness throughout their childhood (Amin-Zaki et al. 1974). Researchers have also observed a heightened incidence of cerebral palsy in children born to mothers in the Minamata Bay (Matsumoto Koya and Takeuchi 1965). Mercury has recently been implicated as being a contributing factor to the increasing prevalence of autism in American children. The Autism Research Institute has focused on mercury containing vaccines (TMS) and their relationship to autism. Over 2 million individuals are affected with autism a neurodevelopment syndrome that typically produces impairment in sociality communication and sensory/perceptual processes and recent evidence has found a positive correlation between complications seen in autistics and complications seen in mercury poisoned individuals (Bernard et al. 2000). While it is difficult to ascribe causation in this case it should not be altogether dismissed. Mercury poisoning has been implicated in the development of many other human dysfunctional states for many years. Among these are cerebral palsy amyotrophic lateral sclerosis Parkinson's disease psychosis and chronic fatigue syndrome (Adams et al. 1983; Bernard et al. 2000; Dales 1972).

We are beginning to understand the threat that heavy metal toxins are to our health. However heavy metal toxicity is a condition that often goes overlooked in traditional medical diagnoses. While it is rare for an individual to experience a disease or health condition solely from a heavy metal toxin it is reasonable to conclude that these toxins exert a dramatic effect on the health of an individual and contribute to the progression of many different debilitating conditions. We have seen how just 5 heavy metals and their respective compounds can adversely affect an individual's health. These effects range from simple gastrointestinal disturbances to severe emotional and cognitive disturbances. Metal toxins have the ability to impair not just a single cell or tissue but many of the body's systems that are responsible for our behavior mental health and proper physiological functioning that we depend on for sustained life. If undetected these agents can cause immeasurable pain and suffering for any afflicted individual. Fortunately there are avenues that an affected individual can pursue to detoxify heavy metals already in their system. Popular therapies (known as chelation) today rely on intravenous (IV) solutions to help eliminate heavy metal toxins. EDTA and DMSA are two compounds that are being used for the removal of heavy metals today. These therapies have been shown to be effective but also potentially harmful to many individuals. Alternative chelation therapies have been developed that are safer than the traditional IV therapies and may prove to be just as effective. These therapies popularly known as oral chelation therapies rely on nutritional substances that have been shown to help detoxify heavy metals within the body and help support the body's overall health.

Oral Chelation II / Age-Less II for Heavy Metal Toxicity and Cardiovascular Conditions

Heavy metal toxicity is frequently the result of long term low level exposure to pollutants common in our environment: air water food and numerous consumer products. Exposure to toxic metals is associated with many chronic diseases. Recent research has found that even low levels of lead

mercury cadmium aluminum and arsenic can cause a wide variety of health problems